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Aspiration pneumonia: A review of modern trends

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ABSTRACT

Purpose: The purpose was to describe aspiration pneumonia in the context of other lung infections and aspiration syndromes and to distinguish between the main scenarios commonly implied when the terms *aspiration* or *aspiration pneumonia* are used. Finally, we aim to summarize current evidence surrounding the diagnosis, microbiology, treatment, risks, and prevention of aspiration pneumonia.
Materials and methods: Medline was searched from inception to November 2013. All descriptive or experimental studies that added to the understanding of aspiration pneumonia were reviewed. All studies that provided insight into the clinical aspiration syndromes, historical context, diagnosis, microbiology, risk factors, prevention, and treatment were summarized within the text.
Results: Despite the original teaching, aspiration pneumonia is difficult to distinguish from other pneumonia syndromes. The microbiology of pneumonia after a macroaspiration has changed over the last 60 years from an anaerobic infection to one of aerobic and nosocomial bacteria. Successful antibiotic therapy has been achieved with several antibiotics. Various risks for aspiration have been described leading to several proposed preventative measures.
Conclusions: Aspiration pneumonia is a disease with a distinct pathophysiology. In the modern era, aspiration pneumonia is rarely solely an anaerobic infection. Antibiotic treatment is largely dependent on the clinical scenario. Several measures may help prevent aspiration pneumonia.

The word *aspiration* simply refers to the drawing in or out of a substance by suction. The term is commonly used in the patient care setting to denote that contents of the oral or upper gastrointestinal tract have passed through the trachea and larynx and entered the lung. The term *aspiration* does not itself indicate the nature of the inoculum or the consequences of the event [1].

Given this broad use of the term *aspiration*, classifying the majority of bacterial pneumonias as a consequence of aspiration is strictly correct based on known pathophysiology of community-acquired (CAP) and hospital-acquired pneumonia (HAP) [2–5]. However, when a clinician uses the term *aspiration pneumonia*, he or she is typically implying a subset of bacterial pneumonia that, although sharing the common pathophysiologic mechanism with most other pneumonias, represents a unique entity of a macroaspiration event resulting in pneumonia. The unique circumstance associated with this term has evolved over time and now may lead to confusion when physicians of different generations interact.

The goal of this review is to describe classic aspiration pneumonias in the greater context of other lung infections and aspiration syndromes. We will attempt to distinguish between the main clinical scenarios commonly implied when the terms *aspiration* or *aspiration pneumonia* are used. We will then review current evidence surrounding its diagnosis, microbiology with implications for treatment, risk factors, and prevention.

1. Common consequences of aspiration

It is important to understand that aspiration is a common event that may lie within the spectrum of normal physiology. A large proportion of healthy people with normal mental status aspirate during sleep based on the detection of radiolabeled oral dyes in the lungs of healthy volunteers [6–8]. The anesthesia literature began highlighting aspiration during ether anesthesia as early as 1950 based on case reports and animal studies carried out during the 19th century [9]. These reports continued with more modern anesthetic agents as well [10,11]. These studies used inert colored dyes ingested roughly 30 minutes before anesthesia and confirmed aspiration with bronchoscopy. These investigators astutely noted that younger, healthier patients almost always tolerated this aspiration without consequence and without respiratory morbidity. This was the first insight into the

Abbreviations: ACEI, angiotensin-converting enzyme inhibitor; AMS, altered mental status; ARDS, acute respiratory distress syndrome; BAL, bronchoalveolar lavage; BO, bronchiolitis obliterans; CAP, community-acquired pneumonia; COPD, chronic obstructive pulmonary disease; GERD, gastroesophageal reflux disease; HAP, hospital-acquired pneumonia; ICD-9, *International Classification of Diseases, Ninth Revision*; MDR, multidrug-resistant; MRSA, methicillin-resistant *Staphylococcus aureus*; RCT, randomized controlled trial; VAP, ventilator-associated pneumonia.

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fact that pneumonia results from a complex interaction between host and inoculum, as opposed to an inoculum alone [12]. Therefore, one of the most common consequences of aspiration is actually to have no consequence—the inoculum is cleared by the normal airway and/or parenchymal host defenses without overt clinical syndromes.

2. Clinical syndromes

Although occurring in otherwise healthy people, several important clinical consequences of aspiration can occur. The most clinically relevant are listed in Table 1. These various manifestations of aspiration can be distinguished by 3 main characteristics—whether the inoculum is infectious or not, the volume of the inoculum, and the acuity of onset of the clinical syndrome.

Many of the aspiration syndromes are a result of noninfectious microaspiration, often due to gastroesophogeal reflux disease (GERD). These include chronic cough syndromes, exacerbation of asthma/bronchospasm, bronchiolitis obliterans (BO) in lung transplant patients, and worsening of chronic fibrotic lung diseases, particularly idiopathic pulmonary fibrosis and systemic sclerosis (scleroderma). Chronic microaspiration itself has also been implicated as a cause of pulmonary fibrosis. The strongest evidence is with microaspiration of exogenous substances such as chronic lipoid pneumonia. Whether chronic microaspiration of refluxed stomach contents alone results in clinically significant pulmonary fibrosis is still unclear. Our review will concentrate on the lower respiratory tract consequences because chronic cough, exacerbation of asthma/bronchospasm, and BO would each require an extensive review [13,14].

2.1. Chemical pneumonitis

Chemical pneumonitis is characterized by macroaspiration of noxious liquids with immediate hypoxemia, fever, tachycardia, and abnormal chest radiograph and lung examination result. The most common noxious fluid is sterile gastric contents, although others such as bile and other agents instilled into the stomach may also result in this syndrome. This specific entity was first described in the anesthesia literature in the late 1940s by Mendelson [15] in a series of women who aspirated during obstetric anesthesia. In Mendelson's series, all 61 young and otherwise healthy patients who aspirated liquid gastric contents recovered within 36 hours with no clear permanent sequelae (5 others aspirated solid material, resulting in 2 deaths by airway obstruction). Subsequently, the wide range of severity from transient hypoxemia to acute respiratory distress syndrome (ARDS) [15–19] has become apparent. Prospective studies of ARDS suggest that 16.5% of patients thought to have experienced aspiration developed ARDS [20]. If ARDS does occur, a particularly severe subtype with a high mortality ensues [18].

Animal experiments helped differentiate the pathophysiology of chemical pneumonitis from subclinical aspiration based on the pH and volume of gastric material needed to stimulate an immediate and severe inflammatory reaction. Based on experiments using human gastric secretions and rabbit lungs, a pH less than 2.4 was required to cause vigorous inflammation. At higher pH, the reaction seen microscopically was more similar to the changes caused by the instillation of water into the lungs [21]. In terms of quantity, experiments inducing chemical pneumonitis in a dog model required 2 mL of hydrochloric acid solution per kilogram to induce the clinical syndrome [22,23]. Similarly, studies done in rabbits by Mendelson required 20 mL of 0.1 mol/L hydrochloric acid per animal [15]. Based on these measurements, an average 70-kg patient would need to aspirate more than 120 mL of gastric contents to induce chemical pneumonitis assuming a gastric pH of 1.

Table 1
Aspiration syndromes

	Infectious inoculum	Acuity of onset	Volume
Airway syndromes			
Chronic cough	No	Chronic	Micro
Exacerbation of asthma/bronchospasm	No	Acute or subacute	Micro
BO in lung transplant	No	Chronic	Micro
Lung parenchymal syndromes			
Exacerbation of fibrotic lung disease	No	Chronic	Micro
Chemical pneumonitis	No	Acute	Macro
Bland aspiration	No	Acute	Macro
Bacterial pneumonia			
Community acquired	Yes	Acute	Micro
Anaerobic pleuropneumonia	Yes	Subacute	Macro
Hospital acquired	Yes	Acute	Variable
Ventilator associated	Yes	Acute	Micro
Aspiration pneumonia	Yes	Acute	Macro

2.2. Bland aspiration

Not all noninfectious macroaspirations cause an inflammatory response in the lung; and therefore, to label these as pneumonitis would be inappropriate. Probably the 2 most common examples are aspiration of blood as a complication of severe epistaxis or hematemesis and the aspiration of enteral feedings. Twenty percent of patients undergoing esophagogastroduodenoscopy will have an infiltrate immediately after the procedure in the dependent lung [24,25]. Most resolve without antibiotic changes. Most episodes of aspiration with enteral nutrition are also uncomplicated [26].

Although bland aspiration may not initially be infectious, blood and enteral feedings represent excellent culture media for growth of either resident bacteria or the small aliquot of bacteria included in the inoculum. Generally, mucociliary clearance and the resident alveolar macrophages can clear the inoculum within hours. The major issue is confusion with an infectious aspiration pneumonia, particularly when the large-volume aspiration is not observed. Prolonged antibiotic treatment is unlikely to prevent this secondary pneumonia but may select for more multidrug-resistant (MDR) pathogens.

2.3. CAP and HAP

Microaspiration has long been known to be the dominant pathophysiologic mechanism behind CAP. Supporting evidence includes the finding that most common CAP-causing microorganisms colonize the oropharynx or nasopharynx in nonhospitalized patients [2,27,28]. Similarly, the pathophysiology underlying HAP, including ventilator-associated pneumonia (VAP), has proved to be microaspiration of oropharyngeal, upper gastrointestinal, or subglottic contents [3,5,29–32]. The distinct microbiology of HAP stems from microaspiration occurring after hospitalized patients become colonized with the virulent organisms found in intensive care unit and hospital environments [4,33–36].

Given the above evidence of aspiration as a common event, development of a parenchymal lung infection depends largely on host defense factors [12,37] and the virulence of the aspirated pathogen. This interaction helps explain the phenomenon of subclinical aspiration without subsequent pneumonia described mostly in young healthy volunteers and surgical candidates.

2.4. Anaerobic pleuropneumonia

Anaerobic pleuropneumonia is probably the entity most commonly meant when the term *aspiration pneumonia* was initially used. Classically, subacute presentation with cough productive of purulent, foul-smelling sputum, and cavitary pneumonia with an associated complicated empyema characterized this syndrome. Patients usually